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Studies on Piglet Mortality

2.—*CLOSTRIDIUM WELCHII* INFECTION*

BY

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Introduction

VARIOUS surveys show that for every 10 piglets born alive eight only survive to weaning ; a mortality rate of 20 per cent. (Tribe, 1954). It is generally accepted that the first 72 hours after farrowing is the most critical period and most of the pre-weaning mortality occurs during this time. It has been customary to attribute most of these early deaths to such factors as crushing, chilling and excessive size of litters, while disease *per se*, has been regarded as being of secondary importance. There is, nevertheless, little accurate information on the relative importance of the various factors concerned in piglet mortality, in particular on the incidence of specific infections, for although surveys are of value in giving an indication of mortality rates, they do not provide precise evidence of the causes of death unless the piglets are subjected to detailed laboratory examination.

It seems probable that in many herds improved standards of management during the last few years have reduced the wastage from such conditions as crushing and chilling, but despite this piglet mortality continues to be a major source of loss to the pig industry. On many farms obscure conditions occur which result in the deaths of whole litters and indeed, on some farms, in the deaths of a succession of litters.

Piglet mortality of this sort has not been adequately investigated in Great Britain and as an initial step, we have attempted to sort out these outbreaks on the bases of the clinical histories and the results of our laboratory examinations. This preliminary study convinced us that we were faced with a complex of problems of varying degrees of importance. The most common cause of mortality appeared to be a condition which clinically resembled the so-called baby pig disease (acute hypoglycaemia in newly-born piglets) as described by Samson, Hester & Graham (1942). In our experience in East Anglia this condition occurred mainly during the late winter and early spring months. Whole litters became affected within 24 hours of birth and generally died within 24 to 36 hours after the appearance of symptoms. On two farms in Hertfordshire, however, although the clinical history had some points in common with this disease, in particular the fact that the piglets were dying within 72 hours of birth, there were certain features which were not in keeping with baby pig disease. On one of the farms mortality was just as heavy in June and July as during the winter months, while

* The first article was published in the *Record* on August 7th, 1954.

piglets on both farms showed certain characteristic differences at *post-mortem* examination which convinced us that we were dealing with a condition which had not been described previously. The present report deals with our investigation into the cause of piglet mortality in these two herds.

Laboratory and Field Investigations

The losses in one of these herds (Farm A) were first brought to our attention in July, 1953, when two piglets were sent to the laboratory for examination and report. An accompanying letter stated that 10 piglets were born alive in the litter during the night of July 8th–9th. Deaths started on the 10th and one piglet only survived on the morning of the 11th, *i.e.*, 48 hours after farrowing. Two of the dead piglets were opened by the owner's veterinary surgeon, who reported that each showed acute inflammation of the small intestines and that all the dead piglets showed signs of blood in the faeces. The two piglets sent to the laboratory showed similar inflammatory changes in the intestines. The report also stated that heavy losses had occurred in 13 of the 33 litters born on the farm during the preceding six to seven months and that in most cases the affected piglets had shown evidence of blood in the faeces.

A few days later three affected piglets from another litter on Farm A were brought to the laboratory. Each of these piglets showed similar acute inflammatory changes in the small intestines.

Cultures prepared from the visceral organs of these five piglets and incubated under aerobic conditions remained bacteriologically sterile but sections prepared from the inflamed portions of the intestines showed extensive necrosis of the intestinal mucosa and invasion of the wall with large numbers of bacilli which morphologically resembled *Cl. welchii* ; findings which suggested the possibility that the deaths were due to *Cl. welchii* infection.

The litters born on Farm A during the next few months remained healthy and the problem was not investigated further until early in March, 1954, when three piglets were received for examination from a litter born on another farm in the same district (Farm B). The veterinary surgeon reported that the losses appeared to be due to the same cause as on Farm A, the piglets dying within 72 hours of birth after showing blood-stained faeces and a distinct reddening of the anus just before death. At *post-mortem* examination each of these piglets showed severe inflammation of the jejunum. It was, in fact, quite striking that this part of the small intestine was severely affected while the duodenum and ileum appeared normal. The contents of the jejunum were reddish brown in colour, while the mucosa in each case showed a velvety appearance. Smears prepared from the intestinal wall showed a mixed bacterial flora, including Gram-positive bacilli which morphologically resembled *Cl. welchii*. The intestinal contents were examined for the presence of toxin. Because of the small quantity of material present in the intestines it was necessary to pool the contents from the three piglets to obtain sufficient material for test. The pooled contents were centrifugalised and 0·2 ml. of the supernatant was injected intravenously into each of two mice. Both mice died within five minutes of injection. A neutralisation test showed that the toxin was inactivated by *Cl. welchii* type C antiserum but not by type D serum. This result indicated that *beta* toxin was present in the intestinal contents. A titration of the supernatant fluid in mice showed that it contained 40 mouse lethal doses in each ml.

Cultures were prepared from the intestines in Robertson's meat broth and tested for toxin after incubation at 37° C. for 18 hours. A toxin was again demonstrated which was neutralised by *Cl. welchii* type C antiserum. Some of the supernatant fluid was trypsinised and, after incubation, tested for *epsilon* toxin. This test proved negative. These results suggested that the organism responsible for the deaths of the piglets was *Cl. welchii* type C. Attempts to isolate the organism from the mixed cultures proved unsuccessful.

A trial was arranged on the two farms in which half of the piglets in each subsequent litter were injected within 12 hours of birth with 2 c.c. of *Cl. welchii* type B antiserum (lamb dysentery serum) which contains *beta* anti-toxin. The injected piglets were earmarked for identification. This injection was well tolerated by the piglets and gave rise to no untoward reactions. In June, two sows, numbers 36 and 20, farrowed on the same day. The subsequent histories of these sows and their litters are as follows.

Sow No. 36

Farrowed six piglets at 6 a.m. on June 22nd. Three piglets were injected with L.D. serum and three left as controls. One control piglet (201) died at 10 a.m. on June 23rd (28 hours after farrowing) and showed some blood-staining of the anus at death. A second control piglet (218) died 48 hours after birth. The rest of the piglets remained healthy. The two dead piglets were sent to the laboratory for examination

Sow No. 20

Farrowed a litter of eight during the night of June 21st–22nd. Four piglets were given L.D. serum. One control piglet (236) became ill when 24 hours old and was killed *in extremis* when 36 hours old. This piglet was sent to the laboratory. Another control piglet developed suggestive symptoms when 72 hours old but subsequently recovered. The injected piglets remained healthy.

On June 25th, *i.e.*, three days after farrowing, rectal swabs and swabs from the skin of the mammary glands and teats were collected from the two sows for culture. The swabs were inoculated on the farm into tubes of Robertson's meat broth.

Laboratory Examination

Piglet No. 201

At *post-mortem* examination the significant lesions were confined to the intestinal tract. The jejunum showed haemorrhagic inflammation, which extended to a lesser degree into the ileum, caecum and colon. The contents of the inflamed areas were reddish brown in colour and the lining of the intestine showed the usual velvety appearance. The stomach was full of partly digested milk. Insufficient contents could be expressed from the intestines for a toxin test in mice but cultures were prepared in Robertson's meat broth medium.

Piglet No. 218

In this piglet inflammation was confined to the jejunum, the lining of which was again diphtheritic giving it a velvety appearance. Sufficient contents were present in the intestines for a mouse inoculation test.

Piglet No. 236

This piglet showed a milder enteritis affecting the jejunum, ileum, caecum and colon. The intestinal contents were blood-stained and present in sufficient quantity to enable a mouse inoculation test to be carried out.

The intestinal contents from piglet 218 were toxic to mice and the toxin was neutralised by *Cl. welchii* type C antiserum. Mice inoculated with intestinal contents from piglet 236 died immediately following injection. No neutralisation occurred with type C antiserum. The deaths were probably due to insufficient clearing of the supernatant or to the presence of decomposition products in the intestinal contents.

All cultures were examined after incubation at 37° C. for 18 hours for the presence of toxin. A toxin which was lethal for mice was demonstrated in the cultures prepared from piglets Nos. 201 and 236, and from both the faecal and mammary gland cultures of sow No. 20. In each case the toxin was neutralised by *Cl. welchii* type C antiserum.

Three cultures, Nos. 201 and 236 isolated from piglets and culture No. 20 isolated from the skin of the sow's udder, were subsequently purified for further study. Pure cultures were obtained by rapid subculture (every two hours) in Robertson's meat broth at 37° C. in a waterbath, followed by plating on 4 per cent. agar containing 10 per cent. horse blood and 1:5,000 chloral hydrate. These three strains were then examined for biochemical reactions and for toxin production. For the latter each strain was grown in 100 ml. of meat broth medium and tested for toxin after 18 hours and again after five days incubation. The biochemical reactions conformed to those generally accepted as being typical of *Cl. welchii*, each strain producing acid and gas in maltose, dextrose, lactose and sucrose. Acid, gas and clot were produced in Litmus milk. No change occurred in mannite, salicin, glycerol and dulcite. A toxin which was lethal for mice was demonstrated in each culture after 18 hours incubation. These toxins were neutralised by *Cl. welchii* types B and C antiserum but not by types A and D antiserum. Similar results were obtained when the cultures were tested after five days incubation. The toxin was destroyed by heating at 60° C. for 30 minutes.

The three strains were subsequently examined at the Wellcome Research Laboratories, through the courtesy of Dr. R. F. Montgomerie. Following routine testing, the strains were reported as being all characteristic *Cl. welchii* type C. Each strain produced *alpha* and *beta* but not *epsilon* toxin.

Histopathology

Sections prepared from various organs showed that the infection was confined to the intestinal tract and mainly to the jejunum. There appeared to be no systemic invasion from the bowel. The liver, spleen and kidney from representative cases showed no significant pathological changes and no bacteria resembling *Cl. welchii* were found in sections stained by Gram's method. Sections prepared from the inflamed areas of intestine all showed extensive necrosis of the mucosa extending into, and in some areas invading the muscularis mucosae. Necrotic tissue, infiltrated and surrounded by masses of gram-positive bacteria, formed a lining to the intestine. These changes were characteristic of all the piglets from which sections were prepared (Figs. 1 to 4). In most cases the adjoining portions of small intestine, together with the whole of the large intestine, showed no pathological changes, and few or no bacteria resembling *Cl. welchii* were seen in sections prepared from these areas.

Clinical History

A complete clinical history was subsequently obtained from the two farms, A and B, which were in different ownership and about two miles apart. No movement of stock had occurred between them, and no sheep had been kept on either farm for at least 25 years. Management in both herds was along conventional lines. Sows and gilts were kept in the open during pregnancy either at pasture or in a wood and were brought into permanent houses about seven days before farrowing. In Herd A, 79 litters were born between December, 1952, and February, 1954, inclusive, and amongst these heavy losses occurred in 20 litters. Of the 197 piglets born alive in the 20 litters, 155 died before they were 72 hours old. Certain changes in management were introduced during 1953 in an attempt to reduce the losses. Some of the sows were allowed to farrow down in huts out-of-doors, while other sows farrowed in pens which had not been used for pigs for at least 10 years. These changes had no significant effect on the mortality rate. Losses were less severe on Farm B. Of 70 litters born between January, 1953, and the end of March, 1954, deaths occurred in 10, with the loss of 47 piglets amongst the 112 born alive. Figures for both farms are given in more detail in Table I. (overleaf).

In both herds piglets appeared quite strong and healthy when born but within 12 to 24 hours of birth some or all of the piglets became dull, showed a disinclination to move around and became rapidly weaker. Some affected piglets died within 30 hours of birth and the majority died within 36 hours ; a few survived up to 72 hours. Examination of dead or dying piglets showed in all cases a reddening of the anus. Most affected piglets showed a red scour in the terminal stages but this was not a constant feature. In most litters no cases occurred after the piglets were 72 hours old but exceptionally a piglet was reported to show symptoms of enteritis at any age up to 14 days following birth. The course of the disease was more prolonged in these cases, affected piglets showing symptoms for several days before dying.

Discussion

Since McEwen (1930) first isolated *Cl. welchii*-type C (*B. paludis*) from cases of struck or strike in adult sheep on the Romney Marsh, infection with this organism has been reported in calves by Griner & Bracken (1953) and in lambs by Griner & Johnson (1954). There are obvious similarities in the age incidence and *post-*

TABLE I
SHOWING INCIDENCE OF LOSSES IN PIGLETS

Month	Farm A				Farm B			
	Number of Litters Born		Affected Litters		Number of Litters Born		Affected Litters	
	Normal	Affected	Piglets Born	Piglets Died	Normal	Affected	Piglets Born	Piglets Died
1952 December	1	1	10	10	—	—	—	—
1953 January	6	1	7	7	3	2	26	14
February	2	1	8	7	1	—	—	—
March	4	3	32	19	4	—	—	—
April	1	—	—	—	5	—	—	—
May	4	—	—	—	1	—	—	—
June	4	1	9	5	5	—	—	—
July	8	7	74	60	8	—	—	—
August	5	—	—	—	8	—	—	—
September	4	—	—	—	4	—	—	—
October	3	—	—	—	2	—	—	—
November	4	—	—	—	3	—	—	—
December	3	1	10	8	4	1	17	4
1954 January	9	4	41	33	5	4	36	17
February	3	1	6	6	—	—	—	—
March	—	—	—	—	7	3	33	12
Totals	59	20	197	155	60	10	112	47

mortem findings in calves, lambs and piglets respectively. Griner & Bracken stated that in calves the greatest loss occurred in animals three to five days old ; infection occurred in lambs 12 to 72 hours old, while in the cases now recorded most of the piglets died within 72 hours of birth. The characteristic *post-mortem* finding in all three species was acute haemorrhagic enteritis with necrosis and desquamation of the mucosa.

Our finding that infection was confined to the small intestine indicates that the disease in piglets is essentially an enterotoxaemia, the bacteria invading and multiplying in the intestinal wall. It seems probable that this is the case also in calves and lambs. The isolation of *Cl. welchii* type C from the faeces and skin of one of the sows gives a clear indication of the probable source of infection. It is reasonable to suppose that the sows ingest the organisms from the soil during the time they are at pasture when pregnant and that as a result they become carriers. When the sows are brought in to pens for farrowing the bacteria are excreted in the faeces and may be present as contaminants on the skin of the teats and mammary glands. The piglets ingest the organisms when sucking and conditions in the intestine of the young animal favour bacterial multiplication and toxin production. The source of infection is probably the same in other species such as calves and lambs.

Griner & Johnson carried out a field trial and showed that the injection of new-born lambs with serum containing *Cl. welchii beta* antitoxin gave complete protection. Furthermore, of 14 sick lambs given 5 c.c. of antitoxin intraperitoneally, nine recovered. While we have not had an opportunity to try the effect of treating affected piglets in this way, the small trial carried out on the two farms suggests that the prophylactic use of serum may be as effective in piglets as in lambs.

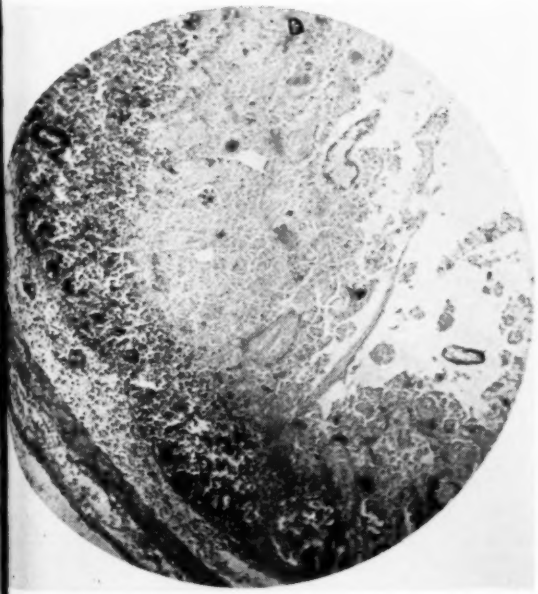
Summary

Heavy mortality in piglets during the first 72 hours of life was found to be due to infection with *Cl. welchii* type C. In addition to isolating *Cl. welchii* in culture from the intestines of affected piglets, the organism was also demonstrated in the faeces and on the skin of one of the sows. The suggestion is made that sows may become carriers of *Cl. welchii* when kept on the land during pregnancy and that the piglets ingest the organisms when sucking. Death follows bacterial multiplication and toxin production in the small intestine, mainly the jejunum. A small field trial suggests that the injection of piglets shortly after birth with a serum containing *Cl. welchii beta* anti-toxin is a useful prophylactic measure.

STUDIES ON PIGLET MORTALITY.—2. *CLOSTRIDIUM WELCHII* INFECTION

E. 1.—Transverse section of small intestine. Stained H and
× 24. Showing extensive necrosis involving the mucosa and
sub-mucosa.

FIG. 2.—Same section as in Fig. 1. Stained Gram × 24. Showing widespread distribution of organisms.



3.—Section of small intestine. Stained H and E × 80.
g extensive necrosis of mucosa, loss of glandular epithelium and massive cellular infiltration.

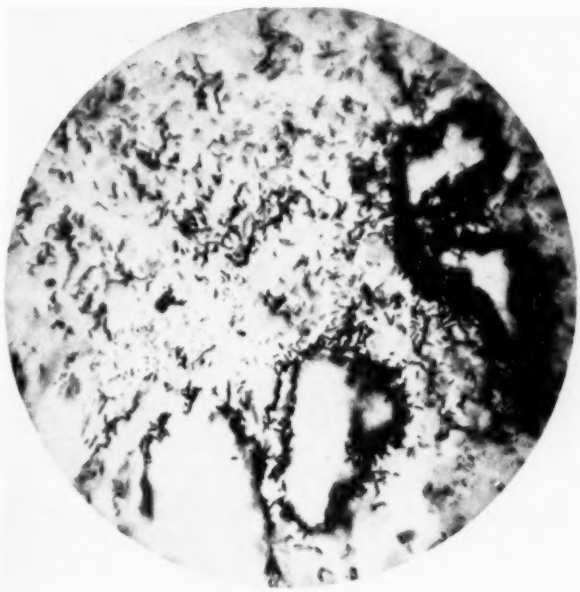


FIG. 4.—Section of necrotic tissue in small intestine. Stained Gram × 350. Showing masses of Gram + organisms.

Addendum

Since this report was prepared for publication, enterotoxaemia has been diagnosed in two piglets received for examination from a farm in Norfolk. These cases showed certain differences from those described in the body of this report. The disease occurred in piglets 7 to 10 days old. Seven out of a litter of nine piglets died during a period of three days. No symptoms were observed by the stockman, the piglets being found dead in the pen.

At *post-mortem* examination the small intestine of one piglet was found to be distended throughout its length. The wall was unusually fragile and the mucosa lightly covered by a yellow fibrinous deposit. The intestinal contents were watery and slightly haemorrhagic. No abnormal changes were noted in the large intestine. There was an excess of peritoneal fluid. The epicardium showed a number of petechial haemorrhages but the other visceral organs showed no pathological changes. The second piglet showed similar changes in the small intestine. In this piglet there was also an area of fibrinous peritonitis which had caused some adjacent loops of small intestine to become adherent. Smears prepared from the intestinal mucosa of each piglet showed the presence of numerous Gram-positive rods which morphologically resembled *Cl. welchii*. A toxin which was lethal for mice and which was neutralised by *Cl. welchii* type C antiserum was demonstrated in the intestinal contents of each piglet. Titrations in mice showed that the intestinal contents of one piglet contained 200 mouse lethal doses of toxin in each ml.; in the second piglet the concentration was 100 m.l.d. A toxin which was also neutralised by *Cl. welchii* type C antiserum was demonstrated in meat broth cultures prepared from the intestinal contents of one of the two piglets. The cultures were tested after incubation for 18 hours. Subsequent examination of the purified culture showed that this was also a strain of *Cl. welchii* type C.

Sections prepared from the small intestines showed a picture similar to that illustrated in Figs. 1 to 4. The mucosa and submucosa showed extensive necrosis and the necrotic tissue was surrounded by large numbers of Gram-positive rods resembling *Cl. welchii*.

The organisms did not appear to be present in such profusion as in the two- to three-day-old piglets but the amount of demonstrable toxin was nevertheless greater. The important difference in the *post-mortem* findings was the absence of haemorrhagic enteritis in the older piglets, the features which suggested the possibility of enterotoxaemia being the diphtheresis of the mucosa and the fluid and slightly haemorrhagic nature of the intestinal contents.

Acknowledgments.—We are indebted to Mr. J. W. Smith and Mr. R. Kimberley, of the Department of Animal Pathology, Cambridge, for the photomicrographs illustrated in Figs. 1 to 4; also to Dr. R. F. Montgomerie, Wellcome Laboratories, Beckenham, for confirming the identity of three strains sent to him for test.

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Note on the Carrier Rate of *L. icterohaemorrhagiae* and *L. canicola* in the Dog

BY

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DURING the routine examination of canine sera for antibodies to *L. icterohaemorrhagiae* and *L. canicola* by the agglutination technique of Schuffner & Mochtar (1927), the incidence of the disease, as shown by positive results, was 41 per cent. This figure was comparable with the observations of earlier workers, such as Stuart (1946), Joshua & Broom (1949) and Broom & Robertson (1951), who found evidence of past or present infection, as indicated by the presence of specific agglutinins, in 40 to 50 per cent. of dogs examined. Broom & MacIntyre (1948), however, reviewing published work, quoted a variation from 12 to 44 per cent. in different parts of the world, while their own observations were 27 per cent.

Seventy unselected canine sera were examined by the same technique to determine the present carrier rate in dogs which had not shown clinical signs of leptospirosis. None of these agglutinated with *L. icterohaemorrhagiae* and only two (2·86 per cent.), with *L. canicola*. All the sera examined were submitted by veterinary surgeons practising in different parts of England.

The results of both groups are shown in Table I.

TABLE I

CANINE SERA EXAMINED FOR LEPTOSPIRAL ANTIBODIES

Serum	No. examined	Total No. positive	Percentage positive	Agglutinins present		
				L. icterohaemorrhagiae	*L. canicola*	Both
Clinical cases	60	25	41·6	1	21	3
Unselected sera	70	2	2·8	0	2	0

The titres of the positive sera were similar in both groups and varied from 1 : 30 to 1 : 100 for *L. icterohaemorrhagiae* and from 1 : 30 to 1 : 1,000 for *L. canicola*.

The above figures suggest that while a similar proportion of clinically suspicious cases of leptospirosis in the dog, namely 41 per cent., were confirmed serologically at the present time, there is apparently a marked decline in the carrier rate, compared with that observed several years ago. The latter has fallen from a minimum level of 27 per cent. as found by Broom & MacIntyre in 1948 to 2·8 per cent. in the dog population studied. A similar decline (to 5·7 per cent.) in the carrier rate has been observed by Hudson (1954) during recent years, but along with it correspondingly fewer clinically suspicious cases (25 per cent.) were confirmed serologically during his investigations.

The apparent discrepancy between the incidence rates found by Hudson and myself might be due to the fact

(Contd. at foot of col. 1 overleaf)

The Semen of the Pig

BY

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IN recent years many pig breeders have become interested in the possibility of artificial insemination in swine, but difficulties are soon encountered in investigations of this subject, and progress towards a solution has been admittedly slow.

Firstly, the survival of boar sperm *in vitro* is far from being as good as that of bull or ram spermatozoa. This precludes the storage of boar sperm under the usual conditions. In addition, the boar is unique among the domestic animals in the enormous volume of semen—relative to body size—which it ejaculates. This volume of semen results from the excessive secretion of the accessory glands, and besides producing an obvious practical difficulty in artificial insemination, may be in part the cause of the poor survival of the spermatozoa contained within it. Until recently, attempts to store pig spermatozoa by freezing at -79° C. did not prove very satisfactory ; it seemed that the sperm failed to survive the period of pre-treatment with glycerol before freezing. Some of the sperm which did survive this period appeared to suffer no ill effects from the freezing and could be kept frozen for a considerable period of time, with recovery of motility on thawing.

McKenzie, Miller & Bauguess (1938) stated that the voluminous seminal plasma of the boar may help to wash the sperm into the sow's uterus. Although it is known that the boar normally deposits the semen directly into the sow's uterus (Berger, 1952), this theory seems consistent with the anatomical features of the uterus of the sow, which has peculiarly long uterine horns. It would seem, therefore, that if successful fertilisation is to be achieved, whole semen may be necessary. On the other hand, Ito, Niwa & Kudo (1948), showed that *artificial* impregnation can occur with a more concentrated sperm suspension and the elimination of much of the natural fluid.

Semen may be described as being analogous to blood in that it consists of a cellular and an acellular portion. The cellular part of the semen is made up mainly of spermatozoa and the acellular part consists of the so-called seminal plasma or accessory secretions. It is generally accepted that spermatozoa were first described by Leeuwenhoek in 1677 after their discovery by Ham, but it was the 19th century that hailed a better understanding of the true nature of spermatozoa. In 1849 Wagner & Leuckart claimed that the spermatozoa "... are merely elementary constituents of the animal organisation like the ova, constituents equally necessary for the spermatic fluid as the blood-globules are for the blood." They did not hesitate to proclaim their belief that spermatozoa were necessary for fertilisation, and Cole (1930), commenting on their work, wrote, "... The nature of the spermatic animalcula having thus been discovered, an approach was open for an attack on the precise rôle which they play in the generative process." Since this time much work has been done in the sphere and many studies on the mechanism of fertilisation have been made.

In contrast, Wagner & Leuckart (1849) considered the *liquor seminis* to be of subordinate significance to the spermatozoa and suggested that it may be merely an incidental element of the semen, the purpose of which was to hold the spermatozoa in suspension. Until recently very little more than this could be said about the seminal plasma of the boar, and it would appear from the form of a boar's ejaculate, that particular attention should be paid to the fluid portion of its semen. The abundance of fluid and the relatively low concentration of spermatozoa are features of boar semen which make it excellent for the study of its chemical composition, and since the more empirical approach of testing various media for the preservation of the sperm *in vitro* initially proved disappointing, the indication for a fundamental analysis of pig semen became apparent.

Collection of Semen

The ejaculate from a pig can be collected by means of an artificial vagina. One such instrument was described by Wallace (1949) and was based on an original design described by Milovanov (1934). The instrument is practically the same as that used for collecting bull or ram semen, except that an artificial "cervix" made of soft rubber is placed inside the cylinder and a pump is attached at the side with which to produce rhythmical pulsations of the rubber lining during the collection procedure. The boar is induced to mount an "artificial sow" consisting of an iron framework covered with sacking. This method is illustrated in Plate I.

The preliminary sexual behaviour or "foreplay" of the boar is the same with the "artificial sow" as it is before normal coitus, consisting of his biting the "sow" on the back and flanks and frequently lifting "her" under the hind legs with his nose. No difficulty is generally experienced in inducing a sexually mature boar to mount such a "dummy sow" although variations in libido are to be expected, and are indeed very

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that many sera sent to us for testing were taken from dogs which had not shown definite clinical signs of leptospirosis, but were submitted as a precautionary measure where a diagnosis had proved difficult to establish. It seems possible that the fall observed in the carrier rate of the disease may well be attributable to the extensive use of antibiotics in treatment during the last few years.

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PLATE 1.—The collection of boar semen with the artificial vagina.

apparent in different boars. Some young boars that may be tried do not seem to have their sexual drive fully developed though they may be producing semen ; these we have considered as sexually immature and found they were better left for a month or two. In every case, however, whatever the experience of the boar, there is a period after the animal has mounted when he exhibits pelvic thrusts prior to his protracted ejaculatory period which itself may be from 3 to 20 minutes. At this stage the operator must be forbearing ! Patience is required and one must never be in a hurry when collecting semen from a boar.

If a boar has his first sexual experience with an inanimate object, such an object often remains the prime stimulus to his sexual activity. This is important in training boars to the artificial vagina, for if an animal has been allowed normal coitus previously he is sometimes more difficult to train. This, however, is by no means a rule because some young boars appear to need normal coitus before they can be persuaded to mount the " artificial sow " and an old boar, experienced in normal service will usually mount the " dummy sow " without much hesitation. In training boars to the artificial vagina, therefore, there is no set rule to follow and the idiosyncrasies of each individual are to be considered, but once a boar has reached the point of orgasm with the artificial vagina there is usually no subsequent difficulty.

We consider the " artificial sow " to be infinitely preferable to a live sow as a stimulus to the boar when the artificial vagina is being used. A live sow may be very nervous of the whole procedure and despite any methods of restraint her inevitable movements will militate against easy collection.

In contrast to those animals with a short ejaculatory period such as bull, ram or rabbit, the boar does not require a well-defined small temperature range in order to release the reflexes of ejaculation. The main stimulus is probably friction. Warm water is not therefore necessary in the artificial vagina when collecting from the boar, although he will often settle quicker if the artificial vagina is warmed before use. Because of the voluminous ejaculate and urethral secretion, lubrication of the rubber lining of the instrument is not required either.

A boar with good libido will readily ejaculate when a simple rubber sheath replaces the artificial vagina. The type of sheath described by McKenzie (1931) and by Ito *et al.* (1948), have both been successfully used at this station. The artificial vagina shown in Plate I tends to be rather cumbersome, and so a lighter type has been designed (Plate II). This is attached to the crate and held in place by a metal bracket. The length of the instrument has been reduced and a polythene collecting bottle used. The results of these modifications are :—

1. The operator has only to squeeze the pump at the side. This enables him to work single-handed without difficulty.

2. The " gel " of the semen does not obstruct the flow into the collecting vial, and

3. The polythene collecting vial is lighter than a glass one and is unbreakable.

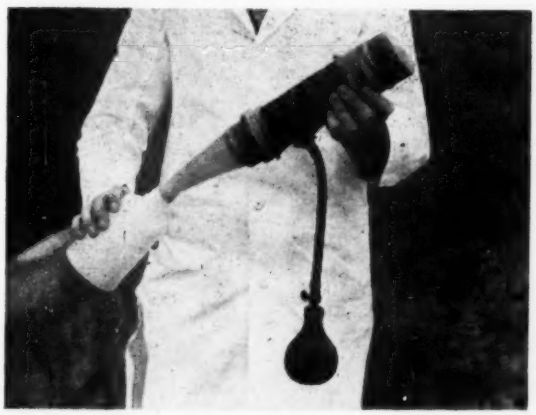

PLATE 2.—The modified type of artificial vagina referred to in the text.

Form of Ejaculate

The volume of semen obtained from the boar at one service may amount to more than 0·5 litre, and according to McKenzie and co-workers (1938) it is ejaculated mostly in two definite waves. Ito *et al.* (1948) classified boar ejaculates into five types, namely, one, two, three or four waves and irregular. In our experience, in addition to the emission of semen at service being divisible into one or more waves, each wave can be separated into fractions on the basis of composition. Like McKenzie *et al.* (1938) we have called these fractions the " pre-sperm," " high-sperm containing " and " post-sperm " fractions. Together, they complete one ejaculatory wave and may be repeated more than once in successive waves.

The pre-sperm fraction of the first wave will often appear yellowish in colour probably due to the presence of urine. The boar has a large preputial pouch which frequently contains stale urine, and this may contaminate and give a characteristic smell to the semen which is

otherwise odourless. The high-sperm fraction is ejaculated next and is immediately followed by an opalescent post-sperm fraction which is poor in sperms but contains much tapioca-like material. The latter is the "gel" the bulk of which is ejaculated at the very end of the wave to complete the post-sperm fraction. The function of the "gel" is said to be to seal up the cervix of the sow and prevent backflow of the semen after coitus.

The ejaculation of the three fractions does not occur in three quite distinct stages; there is considerable mixing of one fraction with another—presumably in the pelvic urethra—so that some gel may be seen in the pre-sperm fraction and indeed throughout the entire ejaculate, and the sperm-rich fraction will contain some vesicular fluid. (See Fig. 2.)

Cellular Elements of the Semen

The wave motion of the spermatozoa such as is seen in the semen of the bull and ram, is not observable in boar semen except on artificial concentration by centrifugation. But with the 1/6 power of the microscope, progressive motility of individual sperm cells can be seen in whole semen and an estimate of the number of such motile cells be made. In this way samples can be graded.

An absorptiometric method of estimating sperm concentration in boar semen cannot be made with accuracy, owing to the presence of opaque matter in the accessory secretions. The best method for such estimations is described by Walton (1927) for rabbit spermatozoa, using the haemocytometer. A dilution of one in 200 with formal-saline is recommended for boar semen. McKenzie and co-workers found no direct relationship between the volume of semen in the boar and sperm density. This is confirmed in Fig. 1, but if this be truly the case, then it must logically ensue that there is a positive relationship between semen volume and the sperm number. It must be emphasised, however, that the number of our observations (Fig. 1) was extremely limited, and cannot really be taken as statistically significant.

Comparatively little is known about the morphology of boar spermatozoa except that they are easily distinguishable from those of other mammalian species by the rather corpulent appearance of the head. Coiled tails, enlarged middle pieces and tailless heads are among the abnormalities which have been observed, as well as the presence of the protoplasmic droplet at various stages of migration. Phillips (1936), found that 62 to 104 abnormal forms of spermatozoa per 1,000 occurred in the semen of normal boars, and stated that sperm morphology was a good indication of fertility. Twenty to fifty per cent. abnormal forms of sperm were present in the semen of boars which produced small litters, weakling piglets, mummified foetuses, or failed to produce conception at all. Milovanov (1936), however, stated that normal boars may have as much as 30 per cent. abnormal forms, and no data are available by which the relationship between abnormal forms and

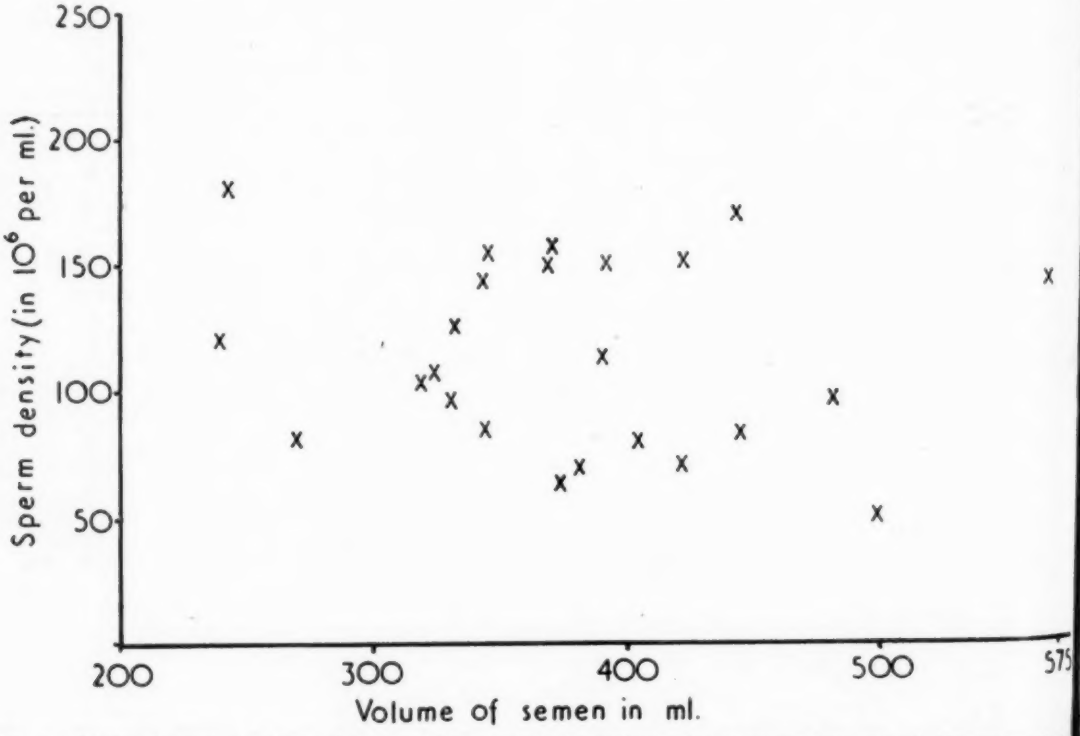

FIG. 1.—Scatter diagram of volume of semen plotted against the concentration of spermatozoa per ml. illustrating that as far as these data were concerned there is no significant relationship between the two criteria.

PLATE 3.—The modified artificial vagina shown attached to the "dummy sow," ready for use.

fertility can be estimated accurately in the pig. Certainly more work on the morphology of pig spermatozoa is required.

Chemical Constituents of the Semen

The normal sugar of semen in all the domestic animals, with the exception of carnivores, is fructose (Mann, 1946, 1948), and it is derived from blood glucose. Fructose can be utilised by the spermatozoon as a substrate for motility both in the presence and absence of oxygen. But in the presence of a small but adequate supply of oxygen, the spermatozoa may oxidise lactic acid, which is the main product of fructolysis (Mann, 1949).

Apart from fructose and lactic acid, the spermatozoa are able to utilise a number of other substances such as glucose, mannose and various fatty acids. Some of these substances may be present in the secretions of the female reproductive tract. This would account for the fact that spermatozoa, even if deprived of seminal plasma, can survive and retain their fertility when placed in the uterus. On the other hand, it must also be remembered that while in the *absence* of oxygen the spermatozoa cannot remain motile without a glycolysable sugar (fructose); aerobically, that is in the

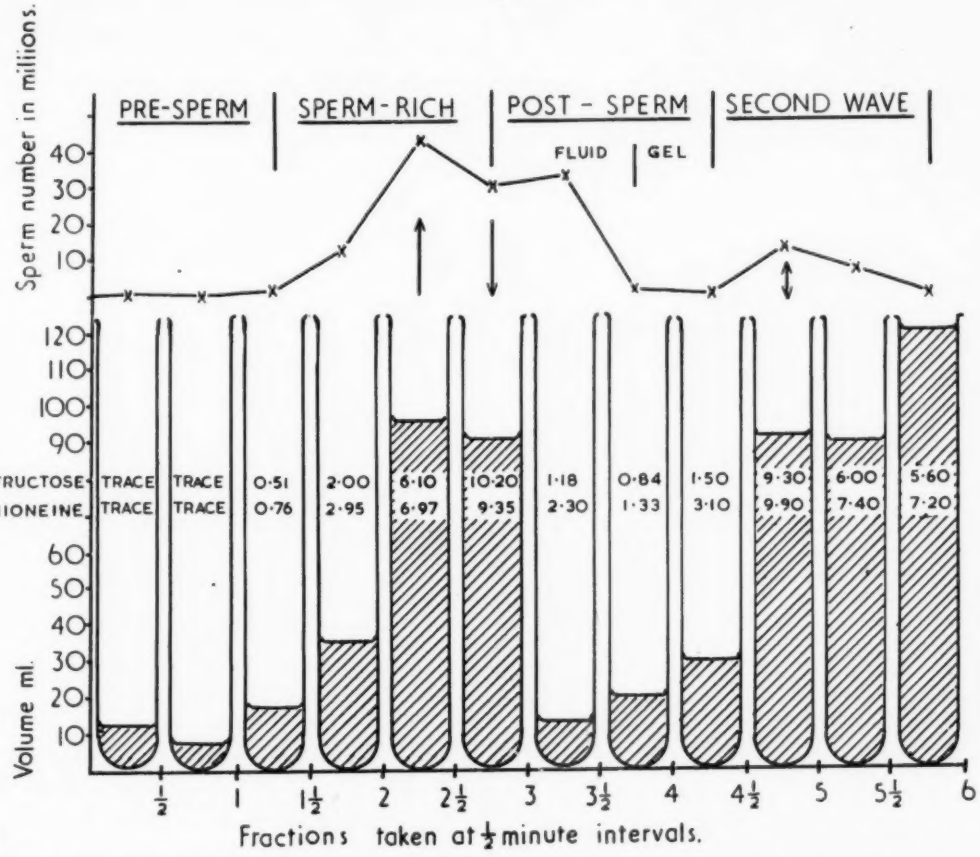

FIG. 2.—Typical ejaculate from the boar, showing the fractions taken at half minute intervals, illustrating the volume, sperm, fructose and ergothioneine contents. It may be noted that in the first wave, the sperm content reaches its peak just before fructose and ergothioneine as emphasised by the arrows, but that there is considerable mixing of these fractions in the final ejaculate.

presence of oxygen, as for example, when in the female tract, the spermatozoa can utilise their own *intracellular* reserve (probably a lipid), and survive at the expense of the metabolic energy derived from the oxidation of this intracellular source.

Hence epididymal spermatozoa which have not come into contact with fructose may be fully active and fertile when placed in the uterus. It is perhaps rather surprising that such abundant seminal vesicle secretion as occurs in boar semen should be so low in fructose particularly when citric acid which is also produced here occurs in such high concentration (Humphrey & Mann, 1949). The exact function of citric acid is obscure, but it is probably involved in the process of gelification.

In addition to these two organic components, electrolytes such as sodium, potassium, calcium, magnesium and chloride, are also present ; these electrolytes vary only slightly in the different fractions of the boar's ejaculate (McKenzie *et al.*, 1938). This is unlike the concentrations of organic materials in the different accessory glands. These vary considerably from one gland to another, and by virtue of this, have been used in the assessment of seminal vesicle function (Mann, 1946 and 1948 ; Mann & Glover, 1954) and to indicate the sequence of events in the process of ejaculation in the boar (Glover & Mann, 1954). Phosphates occur in both organic and inorganic form, the total concentration fluctuating little throughout the ejaculate.

Boar semen has a high reducing value towards dichlorophenol, indophenol and silver nitrate, and this has been found to be due to an organic constituent, namely, ergothioneine (Leone & Mann, 1951 ; Mann & Leone, 1953). This substance is a betaine of thiol-histidine and it was originally recognised in ergot. Its high reducing properties could be of significance in the normal metabolism of pig sperm by virtue of its reducing powers towards sulphydryl groups in spermatozoa. The precise mode of biosynthesis of ergothioneine is as yet unknown, but it is known that the sulphur of methionine is incorporated into seminal ergothioneine, and also that dietary ergothioneine appears in the seminal plasma (Heath, Rimington, Glover, Mann & Leone, 1953).

In 1951, Mann discovered the presence of *meso*-inositol in the seminal plasma of the boar. It is present in high concentration and probably plays a part in maintaining the osmotic pressure of the semen (Mann, 1953). It is interesting to note that Nixon (1952) reported the occurrence of inositol in the amniotic and allantoic fluids of several mammalian foetuses.

It would seem that the " liquor seminis " of boar semen is far from being " merely incidental," that its organic constituents are of the greatest interest and certainly include substances which affect the spermatozoon.

Conclusions

Boar semen is different from that of other domestic animals as regards volume, sperm density, mode of ejaculation and chemical composition ; only the semen of the stallion bears any fairly close resemblance to it. It is possible that the spermatozoon of the boar is physiologically different from that of bull and ram, since boar spermatozoa do not survive well *in vitro* even when freed from the accessory fluids. In view of the unusual chemical composition this must also be taken into consideration in relation to the problems of survival. It remains for fundamental knowledge to be applied to the development and refining of artificial insemination techniques, together with more detailed studies on the morphology and metabolism of the sperm cells in the semen of this species.

Acknowledgments.—With regard to personal experimental work on the semen of the boar, some results of which have been used in this paper, the author is indebted to Dr. Arthur Walton for his help and advice, to Dr. T. Mann, F.R.S., with whom much of the work has been done in collaboration, and to Dr. J. Hammond, F.R.S., for his interest and encouragement. Thanks are due to Drs. Walton & Mann for their valuable criticism of this paper. The author also wishes to acknowledge the kindness of Mr. R. W. Pomeroy in permitting him to use the boars at the Animal Research Station, Cambridge.

The work was performed during the tenure of a Research Scholarship from the Animal Health Trust.

The photographs which appear as Plates I, II and III were kindly taken by Mr. A. V. Guntrip, of the Animal Research Station, Cambridge.

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A Case of Unilateral Pyometritis in the Bitch

BY

A. H. HOGG and H. HOLROYD
Leamington Spa Blackburn

WE believe the following case worthy of record inasmuch as it is unique in our experience. There was nothing unusual in the symptoms shown or in the operation performed for their relief. The latter followed the usual lines (ovariohysterectomy) for a case showing the normal symptoms of pyometritis and the bitch made a good recovery. As the illustration shows, however (Fig. 1), the specimen removed at the operation proved to be a perfect example of the disease affecting apparently only one side of the uterus. In the course of practice we have examined literally hundreds of wombs affected with pyometra both at operation and autopsy yet this is the first of its kind we have encountered. It may serve as a pointer to those interested in the aetiology of this complaint in the bitch. Thus the sterile type of pyometritis is believed to originate through hormonal imbalance but definite information is lacking in regard to the hormone or hormones at fault. Confirmation of this may be found in the variety of glandular preparations used in the attempted medical treatment of the disease.

The case illustrated would appear to suggest unilateral ovarian trouble as being primary in its causation. Does this suggest that in the normal bilateral type both ovaries are always faulty? There would appear to be little doubt of this in another example of the complaint (Fig. 2). In the usual type, however, ovarian disease of the cystic type is not as a rule obvious. We do not infer from this that it is necessary to demonstrate distension of the uterine horn to prove disease of the organ when referring to pyometritis. But as a general rule the greater the distention, *e.g.*, in the normal bilateral case, the greater the toxicity of the case. Moreover this "toxicity" so-called arises often from a fluid which is sterile bacteriologically. A sufficient number of cases might produce some correlation between apparently differing types of ovarian dysfunction and consequent varying of the effect produced on the uterus. Judging from experience in dairy cattle it seems to us unlikely that these varying manifestations of ovaro-uterine disease in the bitch are merely gradations of one particular hormonal dysfunction. It is unreasonable therefore to expect that treatment with any single glandular preparation should act as a panacea. Alternatively, although it may be wise, both financially and medically speaking, to resort to surgery whenever the condition is disclosed, the feeling must surely be prominent that with this disease in particular it is in a sense a confession of failure.

Fig. 1. Unilateral pyometra.

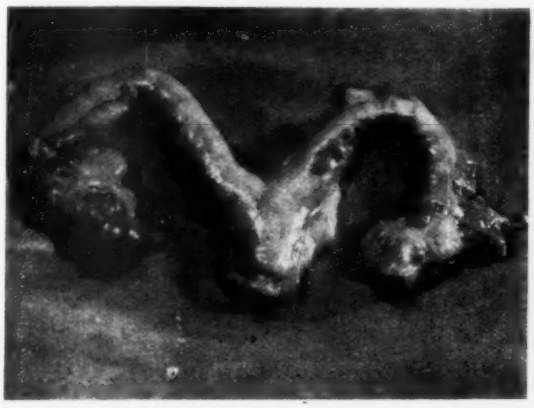

Fig. 2 Pyometra with marked ovarian cysts.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 30th November, 1954		23	—	—	58	—	42
Corresponding period in	1953	24	—	1	37	—	69
	1952	90	—	—	24	—	24
	1951	13	—	37	69	2	61
1st January to 30th November, 1954		303	4	12	717	—	1,336
Corresponding period in	1953	569	—	35	862	—	2,578
	1952	1,051	—	495	230	1	788
	1951	367	—	70	784	8	1,257

TUBERCULOSIS (ATTESTED HERDS) SCHEMES

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at 30th November, 1954, was as follows :—

England	Wales	Scotland	Total (Great Britain)
65,280	31,688	32,929	129,897

Current Literature

DIGEST

Van Den Ende, M. (1954). Our Present Understanding of Viruses. *The Practitioner*, **173.** 525-533. (November, 1954). Four figs., 32 refs.

This article, after defining a virus, emphasises the ubiquity of viruses, the study of which now embraces not only human virus diseases but also the study of bacterial plant, animal and insect viruses. Present knowledge of bacteriophages is reviewed and then follows a review of the present knowledge of animal viruses, particular emphasis being paid to serological and haemagglutination phenomena. In conclusion, it is pointed out that instead of visualising viruses as miniature bacteria it is becoming more appropriate to regard them as relatively complex chemical structures sometimes possessing unique morphological features, in which the essential constituents are protein and nucleic acid. Probably only the latter substance endows viruses with their specific activity, whilst the remaining constituents are but components necessary during their extracellular existence. The majority of pathogenic viruses upset the whole control of normal synthesis by their presence in the cells they occupy. Perhaps, by misdirection of master enzymes viruses create havoc in the normal synthetic process so that the cell is forced to produce virus protein and virus nucleic acid, sometimes with certain characteristic by-products as much foreign to the cell as is the virus. The virus has, in fact, acted as a stimulus for the cell to use its own energy-giving reactions for syntheses which eventually result in its own destruction.

★

Rook, A. J., & Frain-Bell, W. (1954). Cattle Ringworm. *Brit. med. J.*, November 20th. 1198-1200.

This article is a report of observations of cattle ringworm as it affects man. The earliest recorded observations of cattle ringworm in man were made at the end of the 18th century and early in the 19th century.

The nomenclature of the trichophyta still remains confused. It appears that the common cause of cattle ringworm in Great Britain is *Tr. discoides*, a large-spored ectothrix. Although *Tr. mentagrophytes* infections are common in man they are contracted from a wide variety of animals—horses, sheep, dogs, rabbits, mice as well as cattle. There are no reliable statistics as to the incidence of ringworm in cattle in Britain but it is far from uncommon in rural areas. The fungus is known to survive for long periods in the woodwork of buildings, especially in damp, dark surroundings. It is not known whether spontaneous cure at grass is due to exposure to ultra-violet light or to improved nutrition.

A description of the classical lesion of bovine ringworm is given, the normal course lasting six to twelve weeks. Again no reliable figures are available as to the incidence of cattle ringworm in man. Of 39 cases investigated, all except one with an inadequate history had some connection with farm animals, but there is ample evidence of mediate transmission. The incubation period could not be assessed definitely. There was no definite evidence of age or sex susceptibility. The maximal seasonal incidence coincided with the winter months when cattle are housed and the incidence in them is at its highest. The majority of the lesions in man are on exposed areas, particularly the scalp, face, neck and arms. *Tr. discoides* was isolated from all but two of the cases from which *Tr. mentagrophytes* was grown.

The lesion in man is described; this may take four forms. (1) Sharply defined dull-red plaques studded with follicular pustules. (2) An oedematous boggy mass of discharging follicular pustules, the surface of which is often crusted. (3) Erythematous plaques without pustulation. (4) An uncommon type of lesion, irregularly grouped nodules covered by red but unbroken skin.

The more inflammatory lesions are self-limiting in a few weeks, less inflammatory lesions may run a prolonged course. The more inflammatory lesions require only symptomatic treatment, irritating local applications should be avoided. Compresses of potassium permanganate (1:5,000) or mercury perchloride (1:2,000) are helpful. If the lesion is surrounded by an area of cellulitis, a short course of sulphonamide is desirable. The less inflammatory lesions require active treatment. Whitfield's ointment or one of the more recent fungicides should be applied. The risk of producing contact dermatitis with Whitfield's ointment or iodine should be remembered.

There are 15 references.

G. F. B.

(Whitfield's Ointment. Benzoic acid 6 per cent., salicylic acid 3 per cent. in emulsifying ointment.)

News and Comment

R.C.V.S. FELLOWSHIP DIPLOMA AND COLLEGE PRIZES

At the meeting of the Council on January 7th, 1955, the Diploma of Fellowship was presented to Mr. Eric I. Williams for a thesis entitled "A study of reticulo ruminal motility in adult cattle in relation to bloat and traumatic reticulitis with an account of the latter condition as seen in a general practice." Mr. Williams qualified as M.R.C.V.S. in July, 1944, and is in practice at St. Clears in Carmarthenshire.

The Fitzwygram Prize certificate and cheque for £30 were at the same meeting presented to Mr. W. E. Parish and the Williams, Walley and Lawson Prizes certificate and cheque for £20 to Mr. S. A. Hall. The subject for 1953 on which Messrs. Parish and Hall submitted essays was "The day-to-day use of the laboratory in clinical diagnosis."

ELECTION OF MEMBERS OF THE R.C.V.S. COUNCIL

May we draw the attention of our readers to the Notice, which appears just before our classified advertisement columns, formally announcing the procedure of electing five members of the Royal College Council. It has sometimes been suggested in the past that members of the profession are somewhat remiss in casting their votes and show less practical interest in the composition of their governing body than could be desired. It is for this reason that we invite our readers' particular attention to the matter.

UNIVERSITY OF LIVERPOOL

At a Congregation for the conferment of degrees held on December 15th, 1954, the degree of Master of Veterinary Science was conferred on Mr. E. J. H. Ford, B.V.SC., M.R.C.V.S., a member of the staff of the Veterinary Science Division of Boots Pure Drug Company Limited. For his higher degree Mr. Ford investigated certain aspects of Johne's Disease in farm and laboratory animals.

R.C.V.S. EXAMINATIONS

London

Second Examination

Physiology only—Cullinane, J. A.; Davison, R. S.; Jones, W. A.; May, J. N. T.; Pearce, J. S.; Stocks, B. M.

Third Examination

Hygiene only—Baker, K. P.; Barnett, K. C. (credit); Blackmore, D. K. (credit); Bruford, J. C. (credit); Butler, B. C.; Chappell, L. A. (credit); Clark-Lewis, A. V.; Cocks, Edwin (credit); Cook, W. E. R.; Davies, Miss G. M. (credit); Edgar, R. K. (credit); Giles, H. G. (credit); Gill, E. A. (credit); Girling, Miss M. J. (credit); Greenwood, Ronald; Hime, J. M.; Hollands, J. G. (credit); Jones, C. R. V.; Lawrence, B. T.; Mason, J. A.; Mullen, P. A. (credit); Nelson, E. P. (credit); Nettleton, L. B.; Newey, R. M. (credit); Noordin, B. M. K.; Oakley, G. A.; Oke, M. A. (credit); Osman, B. D.; Prasad, A. K. (credit); Ranger, M. J.; Teale, M. L.; Thorp, A. C. P.; Volrath, J. P.; Wells, B. T. (credit).

Pathology only—Davies, M. G.; Fairhall, D. C.; Fenton, B. K.; Gee, R. E.; Gray, C. G.; Hardwick, A. G.; Melluish, H. K.; Millar, J. A.; Mittal, V. P.; Morath, D. E.; Peters, J. B.; Pocock, E. W. (credit); Prole, J. N. B.; Stevens, H. R.; Strang, R. D.; Sutton, J. B.; Watson, Miss D. P.; White, Frederick (credit); Woollett, W. G. W. A.; Wright, M. A.

Final Examination

Alexander, T. J.L.; Arnold, J. H. S.; Booker, J. C.; Cansfield, C. J.; Cawthorne, W. N.; Cullen, C. S.; Davidson, H. M.; Dommett, D. J.; Downes, J. E.; Hambrook, C. L. E.; Holmes, J. R.; Keeling, M. D.; Keep, M. E.; Mears, R. H.; Nelson, D. G.; Nelson, G. J.; Roberts, D. H.; Russell, G. H.; Simpson-White, P. H.; Smith, Miss L. C.; Stalbow, Michael; Thomas, C. J.; Thursby-Pelham, R. H. C.; Tiffin, R. B.; Waller, J. F.; Walter, A. J.

Edinburgh

Third Examination

Beech, J. A.; Hartley, E. G.; Kettles, A. S.; McDougall, G. M.; Moore, T. E.; Nyun, M.; Penman, G.; Smith, P. J.; Stone, M. W.; Stretch, I. L.; Walker, W. D.

Fourth Examination

Baird, J. S.; Cuthbertson, J. L. H.; Gaunt, J. L.; Grant, Forbes; Hovell, G. J. R.; Kay, H. R. M. (Paras.); Kirkbridge, L. M.; Mackenzie, R. M.; Prior, J. G. (Paras.); Shires, R. M.; Williams, G. L.

(Paras.) denotes credit in Parasitology.

Final Examination

Black, Michael; McAllister, Joseph; Stokoe, W. M.; Trainer, P. E.; Watson, A. R. A.

Glasgow

Fourth Examination

Harrison, T. C.

Final Examination

Crawford, T. G.; Gracie, D. K.; Loudon, A. J. B.; Martin, Miss H.; Torrance, D. C.

Bristol

Final Examination

Sebesta, E. G. F.

COMING EVENTS

Jan. 12th.—Meeting of the R.A.V.C. Division, B.V.A., at Boots Pure Drug Co. Veterinary Science Division, The Priory, Thurgarton, Notts, 12 noon.

Jan. 12th.—Meeting of the Supplementary Veterinary Register Division, B.V.A., at the Council Room, R.C.V.S., 9-10, Red Lion Square, London, W.C.1, 1.45 p.m.

Jan. 12th.—Winter Meeting of the North of Scotland Division, B.V.A., in the Northern Hotel, Aberdeen, 2.30 p.m.

Jan. 13th.—Meeting of the Western Counties Division, B.V.A., at the Rougemont Hotel, Exeter, 2 p.m.

Jan. 13th.—Ordinary General Meeting of the South-Eastern Division, B.V.A., at the Royal Star Hotel, Maidstone, 2.30 p.m.

Jan. 21st.—Annual General Meeting of the Mid-West Division, B.V.A., at the Berkeley Café, Clifton, 2.30 p.m.

Jan. 27th.—Annual Dinner-Dance of the North of Scotland Division, B.V.A. (in aid of the Victoria Veterinary Benevolent Fund), in the Northern Hotel, Aberdeen, 7.30 p.m.

Jan. 27th.—Special Meeting of the Hert sand Beds Division, B.V.A., at 70, Holywell Hill, St. Albans, 7.30 p.m.

Jan. 28th.—Annual General Meeting of the Yorkshire Division, B.V.A., in the Hotel Metropole, Leeds, 2.30 p.m.

Feb. 10th.—Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 8.30 p.m.

B.V.A. QUARTERLY MEETINGS

The arrangements for January are:—

Wednesday, January 19th.

10.15 a.m. Organising Committee.
2.0 p.m. Home Appointments Committee.
4.0 p.m. Parliamentary & Public Relations Committee.

At 7, Mansfield Street, W.1.

Thursday, January 20th.

10.30 a.m. Veterinary State Medicine Committee.
2.0 p.m. General Purposes & Finance Committee.

At 7, Mansfield Street, W.1.

Friday, January 21st.

10.30 a.m. Council meeting.
At the Connaught Rooms, Gt. Queen Street.

PERSONAL

Births

BROWN.—On December 30th, 1954, to Marjorie, wife of Robert A. Brown, M.R.C.V.S., of Grove Dene, Preston Road, Yeovil, a sister for Lesley.

MARKHAM.—On November 23rd, 1954, to Joyce (*née* Readings), wife of A. E. G. Markham, B.SC., M.R.C.V.S., of Tanganyika Cattle Ltd., P.O. Iringa, Tanganyika, a daughter, Diana Mary.

METIVIER.—On December 19th, 1954, to Mrs. Hugh Metivier (*née* Irwin), wife of Hugh Metivier, B.A., M.R.C.V.S., of Burleigh Castle, Scarborough, Tobago, B.W.I., a daughter, Michelle MacPherson.

MITCHELL.—On December 20th, 1954, at Griffithstown County Hospital, Monmouthshire, to Sheila (*née* Maclachlan), M.A., and Ken S. Mitchell, M.R.C.V.S., a son, Kenneth Alastair.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax:

Ayr. Hemphill Farm, Galston (Dec. 28).

Durham. Arbour House, Crossgate Moor (Dec. 30).

Lancs. Park Hall Farm, Charnock Richard, Chorley (Dec. 28).

Lincs. Low Farm, Folkingham, Sleaford (Dec. 29).

Northumberland. Burn Mill Farm, Shotley Bridge, Consett; Stone Style Farm, Allendale (Dec. 28).

Wigtown. Barvernochan, Whauphill (Dec. 29).

Yorks. South Clough, Head Farm, Warley, Halifax; Elsingbottom Farm, Farnley, Otley (Dec. 29).

Fowl Pest:

Berks. No. 5, Tileshod Cottages, Clay Hall Lane, Old Windsor (Dec. 29).

Essex. Wrightsbridge Farm, South Weald, Brentwood (Dec. 29).

Hunts. Pig Lane, St. Ives (Dec. 29).

Norfolk. Rose Cottage, Swamp Lane, Gt. Ellingham, Attleborough; 80, Norwich Road, Watton, Thetford (Dec. 28); Marea Farm, School Road, Heacham, Kings Lynn (Dec. 29); 8, Heath Road, Elsing, Dereham; Glen Ayr, Mill Lane, Bradwell, Gt. Yarmouth (Dec. 30).

Swine Fever:

Cardigan. Penrhos, Llangeitho, Tregaron (Dec. 27).

Derbys. The Croft, Fritchley (Dec. 27).

Dunbarton. Edinbarnet Stables, Duntocher, Glasgow (Dec. 30).

Flints. Bryn Coch Farm, Northrop Road, Flint (Dec. 27); Glan-y-Gors, Maeshafn, Llanferres, Mold (Dec. 29).

Glamorgan. St. Julian's Farm, Llanedyrne, Cardiff (Dec. 29).

Kent. St. Anne's, Robin Hood Lane, Bluebell Hill, Aylesford (Jan. 3).

Lancs. Eastfield Farm, Ainsworth Lane, Tonge, Bolton; Halfway House Farm, Church Lane, Lowton, Warrington (Dec. 27); Conder Mill Farm, Scotforth, Lancaster (Dec. 29); Whitley Hall Farm, Greenhall, Wigan; Lower Eccles Hill Farm, Eccles, Darwen; Bridges Farm, Harwood Road, Tottington, Bury (Dec. 30).

Middx. 4, Burrows Hill Close, Stanwell, Staines (Dec. 30).

Montgomery. Park Farm, Groes, Guilsfield, Welshpool; Tyngerddi, Llanfair Caereinion, Welshpool (Dec. 29).

Salop. The Smallholding, Cantlop, Berrington, Shrewsbury (Dec. 29).

Sussex. Steers Farm, Rotherfield, Crowborough (Dec. 29).

Worcs. Little Gains Farm, Cutnall Green, Droitwich (Dec. 29).

OVERSEAS MEMBERS

Members of the Association who will be on leave in this country during the summer are requested to notify the General Secretary, B.V.A., No. 7, Mansfield Street, London, W.1. This is in order that they may receive an invitation to attend a meeting of the B.V.A. Overseas Committee to be held on Wednesday, June 22nd, 1955, if there are any subjects on the agenda in which it is thought they may be interested.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

A LEGITIMATE GRIEVANCE

Sir,—As the Superintendent of an Animals' Refuge in the north of England may I say that I had not realised, until reading in your issue of December 25th the letters of Messrs. McCormick and Hoddinott, the injustice to private practitioners in not being allowed to advertise, while some of those who are attached to free clinic organisations are apparently aiding such organisations in their advertising.

Surely the Royal College will now see that the profession will be freed to advertise as it thinks necessary? If the position is allowed to remain as it is it will breed nothing but bad blood, and any such tension is the last thing to be desired.

Yours faithfully,

ALFRED BRISCO,
Secretary,

The National Equine Defence League,
Carlisle.

Sir,—Much space in THE VETERINARY RECORD has been devoted to the expression of opinions on this matter, I will, therefore, be as brief as possible if I may be permitted to make a few comments in an endeavour to restore a sense of proportion to the subject.

The need for charitable animal clinics does undoubtedly exist, for there are poor people in this country to-day. In general they do not represent the same section of the community as formerly, but their need is neverthless genuine. Every honest veterinary surgeon must admit to a knowledge of persons who have to budget to within a few shillings a week. They can afford occasional private treatment for their animals but not prolonged and expensive treatment. If these people are to be refused charitable treatment (and they are those who suffer most from taxation and the present economic system) they will be discouraged from keeping pets. Any action which tends to decrease the pet population must be to the disadvantage of the veterinary profession. Other professions recognise this poverty; the legal profession, for example, is now considering a proposal to extend the scope of legal aid.

With regard to almoning, unfortunately this can never be 100 per cent. efficient, but the veterinary surgeon attending the clinic is entitled to refuse treatment if he is satisfied that the owner of the animal can afford private treatment. He, therefore, has only himself to blame if the clinic is abused. If cases do arise where the lay staff are exceeding their duties in the veterinary surgeon's absence, we have our remedy under the terms of the 1948 Act and prompt and vigorous measures should be taken.

Concerning Dr. Hare's letter in the issue of November 27th, there is nothing unethical, by professional standards or any other criteria, in working for a firm or institution which advertises. To suggest penalising anyone for so doing is ridiculous, for the professional employees of the drug houses, many of which have been responsible for great advances in medical and veterinary treatments, and without whose resources research would be greatly handicapped, would come under this censure. If this suggestion were carried to its logical conclusion, all members on the staff of the Royal Veterinary College would have to be penalised, for this institution advertises its wares weekly in THE VETERINARY RECORD. This suggested "remedy," if carried out, would also be greatly to the disadvantage of the profession.

Charitable organisations for the welfare of animals are firmly established in the community and our wisest policy is one of co-operation with them. In this way we are better able to observe their activities and to expect a spirit of reciprocal co-operation on their part.

Before any action against the charitable organisations is decided upon, as many facts and figures as possible should be collected and considered (would it be too great a task to find out just how many practices have actually suffered adversely from the proximity of a clinic?) and more unbiased thought should be given to the subject than has been in evidence in some of the correspondence in THE VETERINARY RECORD.

Yours faithfully,

W. M. TAYLOR.

15, Glendower Place,
South Kensington,
London, S.W.7.

January 1st, 1955.

Sir,—Recently I have read with interest the correspondence under the above heading. May I, as one who worked many years for an animal welfare society, express my view?

People visiting these "free clinics" are of three types: The genuinely poor who give until it really hurts—often more than they would be asked to pay by a private practitioner; well-meaning folk who subscribe substantially thinking they are helping animals; wealthy people who want something for nothing.

It is the latter who should be obliged to pay for the treatment of their animals, and should be sent to a private practitioner. One has only to visit one of the hospitals of an animal welfare society, as I did a short time ago, to be amazed at the expensive cars of well-dressed people waiting for treatment for their animals. I counted no fewer than 15 cars and taxis outside these premises.

In the waiting room a notice was displayed which read to this effect: "If you can pay for the services of a private practitioner and come to us you are robbing the poor for whom this dispensary is open. Our staff have instructions to refuse to treat the animal of any person whom they think could pay for the services of a private practitioner."

Is anyone refused on this ground? True there is a book somewhere in the surgery which is occasionally signed by members of the public, which is headed: "I the undersigned am unable to pay for the services of a private practitioner," etc. This is often signed without being read and stands for nothing other than a means of saving the bother of suitable almoning. I would venture to suggest that not more than 10 per cent. sign this book. Surely it should be checked by an independent visiting panel and compared with the daily case book?

Recently several cases have been brought to my notice of people who own houses, cars, television sets, and so on, having their animals accepted for treatment in spite of my protests. To-day I heard of an animal welfare employee collecting cats for conveyance to his society's free hospital for spaying.

It is regrettable that such spaying operations are performed by members of the profession in the employment of certain societies. Surely one would expect something better from members of the Royal College of Veterinary Surgeons?

Yours faithfully,

W. SHEPHERD,

168, Gipsy Road,
Welling, Kent.

January 1st, 1955.

EQUINE RADIOGRAPHY

Sir,—Your general article "Some Practical Aspects of the Experimental Radiography of the Larger Domestic Animals" (*Vet. Rec.* **66.** 593), was of particular interest to me.

I have lived long enough to be only mildly surprised when I find that honour is not always given where honour is due.

In 1939 after some years of labour and expense I completed "A Radiographic Anatomy of the Normal Horse." This was to have been published by Messrs Ballière, Tindall & Cox, but the last war intervened and, alas, the MSS. and radiographs were destroyed by enemy action whilst in their vaults in London. Not only the equine head (which was carefully labelled with every anatomical detail and submitted for Fellowship honours), but all the limbs and all the joints, the thorax and pelvis, the cervical and dorsal spines were clearly portrayed with notes as to positioning, radiographic data and technique and anatomical points.

The "not up to standard" radiographs and technique I retained and only this year despatched them to Professor McCunn, who expressed pleasure at their receipt: "they and the information would be useful to his chaps who were doing X-ray work on horses and goats."

The references at the end of the article give no acknowledgment of my labours 15 years ago.

Yours faithfully,

V. BERWYN JONES,

Frocester Lodge,
Nr. Stonehouse, Glos.

October 3rd, 1954.

PULPY KIDNEY VACCINE

Sir,—Recently we have had an unfortunate experience following the use of Pulpy Kidney Vaccine. A number of sheep have died and a great many have developed abscesses at the site of injection. It is thought that other members may have had similar experiences. It is felt that it will be of considerable help in determining the cause if these members will be so kind as to write to us giving details of such experiences and the results of their investigations.

Yours faithfully,

T. J. MORGAN,
J. H. McGHEE,

46, Coten End,
Warwick.

December 15th, 1954.

IDENTIFICATION OF HORSES

Sir,—I read with interest the report of the sub-committee on the preparation of a system of description of colours and markings of horses. The object of the system is the correct identification of valuable horses, particularly when the identity of two horses is confused. I am wondering if the sub-committee has investigated the use of the chestnuts as a means of identification?

When in America last year, I had the good fortune to be shown the American race-course system of thoroughbred identification by Dr. Woodcock and other veterinary surgeons responsible for the Belmont and Aqueduct tracks. They showed me that their sets of four photographs, one photograph of each chestnut of any horse in question, together with a card index similar to the one shown in the back of the sub-committee's report, provided a system of identification comparable in accuracy with the finger-print system used in human identification.

Each horse is identified by means of this card index and sets of chestnut photographs on being brought to the course prior to a race.

While this may be unduly elaborate for the certification of non-pedigree horses, would it not obviate the possibility of substitution in thoroughbreds?

Yours faithfully,

D. M. BERRY.

1, The Green,
Dinton, Bucks.

December 31st, 1954.

ERRATUM

In Mr. Smythe's letter on parturition in the pig (Dec. 25th) a transposition of wording occurred in the third paragraph. The opening lines should have read: "In the sow, the chorion is laid down over the *whole* of the endometrium, commencing at about the 10th to 15th day of pregnancy and is followed by deposition of the allantois, from the hinder gut, at the third week. The amnion then develops and surrounds the embryo," etc.